

association is influenced by the way in which the control group is selected and in this regard we question the nature of controls studied by Jensen and colleagues. It appears that none of the asymptomatic control subjects was examined microscopically to determine the existence of urethral polymorphonuclear (PMN) leucocytes. Indeed, men with "asymptomatic" urethritis may have been included in the control group. Few investigators have compared the prevalence of mycoplasmas in men with microscopic urethritis who have no signs or symptoms with that in men without urethritis. However, Swartz *et al*<sup>6</sup> found that *Chlamydia trachomatis* was isolated more frequently from men with asymptomatic NGU than from those without objective urethritis, suggesting that urogenital pathogens may be involved in the aetiology of this condition. In addition, it is unclear whether Jensen and colleagues examined the asymptomatic subjects clinically at enrolment. Clearly, asymptomatic men with a discharge on examination and objective urethritis ( $\geq 5$  PMN leucocytes/high-power microscopic field) have "clinical urethritis" and should be excluded from the control group and included in the study group. Inclusion of asymptomatic men who have objective urethritis, with or without an observable discharge, in the control group would prevent proper evaluation of negative associations. This may have influenced to some extent the significance given to *M genitalium* by Jensen and colleagues and biased their results against detecting an association of *U urealyticum* with NGU.

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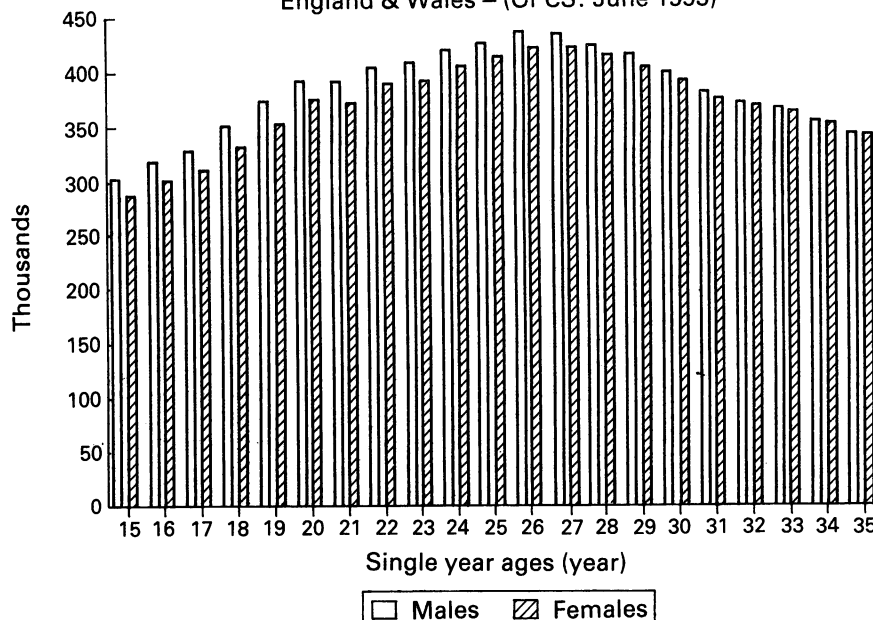
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- 2 Taylor-Robinson D, Furr PM, Tully JG, Barile MF, Møller BR. Animal models of *Mycoplasma genitalium* urogenital infection. *Isr J Med Sci* 1987;23:561-4.
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#### ***Neisseria gonorrhoeae* isolates at St Mary's Hospital, London 1980-91**

Renton *et al*<sup>1</sup> report an increase in isolates of gonorrhoeae at St Mary's in 1990—and a similar pattern has been reported nationally—accompanied by speculation that this may be an indicator of varying success in educational programmes in safe sex. We feel that, before elaborate behavioural explanations are pursued, it must be clarified that demographic factors would in any case suggest a peaking of multiple-partner sexual activity around 1989 and 1990 for England & Wales as a whole.

We attach the latest single year popula-

Final (unrevised) 1991 Population Estimates  
England & Wales – (OPCS: June 1993)



tion estimates for 1991 between the ages 15 and 35 years. The peak in the mid-20s is very marked—and will be still more acute in the revised estimates due shortly. Johnson *et al*<sup>2</sup> identified clear age differences in reported heterosexual multiple-partner sexual activity—although even their sample size (18 876) did not permit calculation of rates for single ages. Nevertheless, an equivalent peaking in the mid-20s may be inferred. As might be expected, attendance at STD clinics was strongly associated with reported numbers of sexual partners—both homosexual and heterosexual. A coincidence of the two peaks in 1990 might well generate the national pattern observed.

From this perspective, it is the dramatic

fall in isolates throughout the 1980s that requires special explanation in terms of changing sexual practices—while the 1990 peak could be partly the reassertion of a demographic secular pattern, as observed in the 50 years up to 1975.

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- 1 Renton AM, Ison CA, Whitaker L, Kirtland K, Kúpak E, Harné JRW. *Neisseria gonorrhoeae* isolated at St Mary's Hospital, London 1990-91. *Genitourin Med* 1991;69:286-9.
- 2 Johnson AM, Wadsworth J, Wallings K, Bradshaw S, Field J. Sexual lifestyles and HIV risk. *Nature* 1992;360:410-2.

## BOOK REVIEWS

**The Human Herpes Viruses.** Edited by Bernard Roizman, Richard J Whitely and Carlos Lopez. New York, Raven Press. (US \$113.50, pp 447.) 1993. ISBN 0-7817-0024-8.

Over the past 25 years there have been several major advances in human herpes viruses research. Firstly, there has been an appreciation of the diversity of clinical diseases caused by this group of viruses, ranging from the trivial to the life-threatening; and the list is getting longer all the time. Second was the recognition that this group of viruses have evolved a variety of complex mechanisms (many of which are still poorly understood) for persisting in the host, probably for life. Third, there has been an appreciation of the vast and complex diversity of this group of viruses. And finally, there has been the recent introduction of safe and effective drugs to treat some of these infections.

The names Roizman, Whitely and Lopez are inextricably linked with human herpes

viruses and the publication of this volume reflects the editors' research commitments, clinical expertise and above all, enthusiasm for the subject. The volume comprises 16 chapters dealing with basic virology, epidemiology, clinical syndromes, anti-viral therapy, immunity and vaccine development. All 16 chapters are well written and readable, although several chapters, notably the chapter on anti-viral therapy, have a paucity of references (including several key publications) from European journals.

I was a little disappointed with the chapter *The Epidemiology and Clinical Manifestations of Herpes Simplex Virus Infection* which did not provide any information concerning the risk of acquisition of either orolabial or genital herpes from symptomatic and asymptomatic sources. In addition, the overview of HSV seroepidemiology was uncritical and referred only to the type specific assays developed in Atlanta, not even mentioning the Western blot type specific assays developed in Seattle.

The most readable chapters, which bring together a good deal of useful and diverse information, are chapter 2 *Herpes Simplex Viruses and their Replication*, chapter 9 *Human Herpes Viruses 6 and 7*; *Molecular Biology and Clinical Aspects*, chapter 10 *The*